Association of Short Sleep Duration with Weight Gain and Obesity at 1-Year Follow-Up: A Large-Scale Prospective Study

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Study Objectives: To investigate the association between short sleep duration and elevated body mass index (BMI) and obesity in a large sample of Japanese adults over a short period

Design: Prospective design with baseline in 2006 and 1-year follow-up

Setting: Workplaces of an electric power company in Japan

Participants: 35,247 company employees (31,477 men, 3,770 women) distributed throughout Japan

Measurements and Results: Measured weight and height and self-reported sleep duration were obtained at annual health checkup in 2006 and 2007. Weight change was defined as the difference in body mass index (BMI) between the baseline and 1 year later. Relative to the reference category (sleep duration 7-8 h), short sleep duration (< 5 and 5-6 h) and long sleep duration ≥ 9 h were associated with an increased risk of weight gain among men after adjustment for covariates. Of the non-obese (BMI < 25) men at baseline, 5.8% became obese (BMI ≥ 25) 1 year later. Higher incidence of obesity was observed among the groups with shorter sleep duration. Adjusted odds ratios for the development of obesity were 1.91 (95%CI 1.36, 2.67) and 1.50 (95%CI 1.24, 1.80) in men who slept < 5 and 5-6 h, respectively. No significant association between sleep duration and weight gain or obesity was found for women.

Conclusions: Short sleep duration was associated with weight gain and the development of obesity over 1 year in men, but not in women.

Keywords: Sleep duration, obesity, body mass index, weight gain, occupational health

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THE PROPORTION OF PEOPLE WITH OBESITY HAS INCREASED GLOBALLY, WITH AT LEAST 1.1 BILLION ADULTS NOW CLASSIFIED AS OVERWEIGHT (DEFINED as a body mass index [BMI] of 25 to 29.9 kg/m^2) or obese (BMI ≥ 30 kg/m^2).[1] The prevalence of overweight and obesity in many Asian countries is around 30%.[2] In particular, prevalence in Japanese men aged 30 to 60 increased from about 20% in 1986 to more than 30% in 2006.[3]

The World Health Organization (WHO) describes obesity as one of the most visible yet neglected public-health problems, which threatens to overwhelm both more and less developed countries.[4] Excess body weight is the sixth most important risk factor contributing to the overall burden of disease worldwide.[5] Obesity has more recently been shown to decrease life expectancy by 7 years at the age of 40 years.[6] Further, the risks of diabetes, hypertension, and dyslipidemia increase from a BMI of about 21.0 kg/m^2, thereby reducing life expectancy and greatly increasing health and societal economic burden.[7]

Factors in the development of obesity or overweight are genetic background, physical inactivity, and the consumption of foods with high energy density, namely those rich in fats, extracted sugars, and refined starches.[8,9] More recently, particular attention has been directed to the potential role of sleep duration as an obesity risk factor. A number of cross-sectional studies have shown that short sleep duration is associated with weight gain in children and adults.[10,11] By definition, however, the findings of these studies give no information about the direction of causality.

Longitudinal research to overcome the problems inherent in the cross-sectional studies is really important, but the results for the association between sleep duration and obesity are not consistent.[12-17] The generalizability of the findings is also hampered by several methodological problems, such as the use of self-reported body weight, selected participants of women only or those at high risk of psychiatric disorders, and small sample size.[12-17] In addition, the follow-up periods in these studies are ≥ 5 years, and thus it is not known whether the prospective association between sleep duration and obesity would be observed within a shorter period of the follow-up. This information may be essential to initiate preventive intervention for sleep-related obesity at an earlier stage.

In the present study, we investigated the association of sleep duration with weight gain and the development of obesity at 1-year follow-up among over 35,000 male and female workers in Japan.

METHODS

Participants

Participants were employees of an electric power company distributed in 12 of 47 prefectures throughout Japan. Baseline data were collected using a self-administered questionnaire during an annual health check-up in 2006 conducted in accordance with the Industrial Safety and Health Law of Japan.

A commentary on this paper appears in this issue on page 143.

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For follow-up, a second self-administered questionnaire was administered at the next health checkup 1 year later. Answers to the questionnaire and health-check up data for 2006 and 2007 were obtained from 35,247 employees (31,477 men, 3,770 women). Participants, except shift workers (n = 3044, 8.7%), generally worked on weekdays and none worked on weekends on a permanent basis. Permission for the study was obtained from the company, and the study protocol was reviewed and approved by the Kitasato University Ethics Committee.

Assessment of Sleep Duration

Usual sleep duration on weekdays was defined as the response to the question, "How many hours do you sleep on weekdays (workdays)?" and that on the weekend as the response to "How many hours do you sleep on the weekend (non-workdays)." Usual daily sleep duration was calculated as the weighted average of weekday and weekend sleep durations using the formula: ([{usual workday sleep duration} × 5] + [{usual weekend sleep duration} × 2])/7.[18] The calculated sleep duration was then classified into 6 categories: < 5 h, 5 to < 6 h, 6 to < 7 h, 7 to < 8 h, 8 to < 9 h, and ≥ 9 h.

Assessment of Body Mass Index and Obesity

Measured height and weight data were obtained from the firm's annual health check-up records. Body weight was measured to the accuracy of 0.1 kg on participants wearing light clothes and without shoes with digital scale measurement that calibrated before use against a standard weight by trained people. Height was also measured without shoes with both digital and analog scale measurements by trained people. Body mass index (BMI) was calculated as weight in kilograms divided by height in meters squared. Although the WHO defines obesity as a BMI ≥ 30,[4] the Regional Office for the Western Pacific Region of WHO, the International Association for the Study of Obesity and the International Obesity Task Force have proposed that adult obesity in Asians be specified as a BMI ≥ 25 (WPRO criteria).[19] We used this criterion to define obesity. Two outcomes were assessed, namely the difference between BMI at baseline and 1 year later, and the development of clinical obesity as defined above.

Covariates

The questionnaire inquired about potential confounding factors, namely age (in 1-year strata), alcohol consumption (ethanol g/week) and physical activity (weighted average of walking hours on weekdays and weekends) as continuous variables, and gender, rotating 8-h shift work (yes or no), habit of smoking (yes or no), and depressive symptoms (yes or no) as categorical variables. Physical activity on weekdays was defined as the response to the question, "How many hours do you walk on an average a day on weekdays (workdays)?" and that on the weekend as the response to "How many hours do you walk on an average on weekend (non-workdays)? Usual physical activity was calculated as a weighted average of weekday and weekend walking hours a week, using the formula: ([usual weekday walking hours] × 5) + ([usual weekend walking hours] × 2). The presence of depressive symptoms was established by a positive response to either of the two questions on "depressive mood" and "loss of interest or pleasure" in the Mini-International Neuropsychiatric Interview (MINI), a short structured interview of the Structured Clinical Interview for DSM- IV.[20,21]

Statistical Analysis

All analyses were performed separately for men and women. Baseline characteristics are presented as mean ± standard deviation (SD) or numbers (percentages). Statistical differences in categorical variables and in continuous variables between the 6 categories of sleep duration at baseline were determined using the χ^2 test and one-way analysis of variance (ANOVA), respectively. When results for ANOVA were significant, the Dunnet test was used to test the associations between categories of sleep duration and the continuous variables at baseline, with values in the group sleeping 7-8 h used as reference. Associations between categories of sleep duration and continuous measures of BMI gain were tested using multivariable linear regression analyses with adjustment for age, BMI at baseline, shift workers, smoking, alcohol consumption, physical activity, and depressive symptoms. Multivariable logistic regression analyses were conducted to examine the association between the development of obesity and each sleep duration category relative to the 7-8 h referent group. Baseline covariates for adjustment included age, shift workers, smoking, alcohol consumption, physical activity, and depressive symptoms. All analyses were conducted using SPSS software version 12J for Windows (SPSS, Inc.; Chicago, IL, USA).

RESULTS

Of 35,247 participants, 395 (1.1%) were excluded because of missing data (362 for BMI data, 23 for work schedule, 7 for depressive symptoms, 2 for physical activity, and 1 for alcohol consumption), leaving 34,852 (31,206 men, 3,646 women) for analysis. The excluded employees were older (45.3 years ± SD 8.3 vs 39.8 ± 9.6, $p < 0.001$), had more sleep time (6.7 h ± SD 1.1 vs 6.5 ± 1.0, $p < 0.01$), were more likely to be women (31.4% vs 10.5%, $p < 0.001$), had less physical exercise (10.8 walking h/week ± SD 10.8 vs 11.8 ± 10.3, $p = 0.04$), were less likely to be smokers (29.4% vs 38.4%, $p < 0.001$), and had no significant differences in shift work, alcohol consumption, and depressive symptoms when compared to the participants analyzed. Characteristics of men and women categorized by sleep duration at baseline are shown in Table 1. Mean age (SD) was 40.0 (9.6) and 38.0 years (9.0), respectively. Of the total, 11,183 (35.8%) men and 457 (12.5%) women were obese. Average sleep duration (SD) was 6.6 (0.9) and 6.4 h (1.0), respectively. Almost half of the participants slept for 6-7 h. On cross-sectional analysis, BMI was significantly greater for participants with sleep duration of less than 5, 5-6, and 6-7 h than those with 7-8 h among both men and women.

Table 2 shows the prospective association between sleep duration and BMI gain. After adjustment for covariates, multivariable linear regression analysis showed a significant association between BMI gain and sleep duration < 5 h ($\beta = 0.016$, 95% confidence interval (CI) 0.024, 0.146), 5-6 h ($\beta = 0.013$, 95%CI 0.001, 0.061), and ≥ 9 h ($\beta = 0.018$, 95%CI 0.079, 0.340) relative to 7-8 h among men. Among women, in contrast, no as-

Table 1—Baseline characteristics of the participants by sleep duration category by sex

	Sleep Duration (Hours)						
	< 5	5 to < 6	6 to < 7	7 to < 8	8 to < 9	≥ 9	P value
Men							
n	811	4716	15262	8677	1576	164	
Age, years	39.4 (8.9)	39 (8.7)	39.6 (9.5)	40.6 (10.1)	42.3 (10.4)	42 (11.2)	< 0.001
Body mass index (BMI), kg/m^2	24.4 (3.7)	24.1 (3.4)	23.8 (3.3)	23.6 (3.2)	23.6 (3.1)	23.5 (3.5)	< 0.001
BMI gain†, kg/m^2							
Median	0.07	0.05	0.03	0.00	−0.04	0.07	
Interquartile Range							
1st Quartile	−7.42, −0.45	−6.52, −0.44	−7.76, −0.44	−6.41, −0.44	−7.97, −0.50	−1.80, −0.23	
2nd Quartile	−0.44, 0.07	−0.44, 0.05	−0.44, 0.03	−0.44, 0.00	−0.49, −0.04	−0.23, 0.07	
3rd Quartile	0.07, 0.58	0.05, 0.54	0.03, 0.47	0.00, 0.45	−0.04, 0.42	0.07, 0.56	
4th Quartile	0.59, 8.04	0.54, 5.51	0.47, 6.90	0.45, 5.37	0.43, 3.08	0.57, 6.58	
Obesity‡, n (%)	355 (43.8)	1873 (39.7)	5478 (35.9)	2889 (33.3)	533 (33.8)	55 (33.5)	< 0.001
Smokers, n (%)	346 (42.7)	1837 (39)	6107 (40)	3938 (45.4)	799 (50.7)	92 (56.1)	< 0.001
Alcohol consumption, ethanol g/week	89.5 (122.4)	95.6 (121.4)	106 (122.2)	123.9 (133.7)	143.8 (146)	145.3 (152)	< 0.001
Walking hours a week, hours/week	12.3 (10.1)	11.5 (9.6)	12.1 (10)	11.8 (9.9)	12.1 (10.8)	12.7 (13.8)	0.01
Shift workers, n (%)	66 (8.1)	314 (6.7)	1270 (8.3)	966 (11.1)	240 (15.2)	35 (21.3)	< 0.001
Depressive symptoms, n (%)	54 (6.7)	180 (3.8)	344 (2.3)	202 (2.3)	43 (2.7)	8 (4.9)	< 0.001
Women							
n	131	760	1764	831	145	15	
Age, years	41.5 (8.9)	39.7 (8.9)	38.3 (9.5)	35.8 (8.9)	35.4 (8.3)	36.3 (10.3)	< 0.001
Body mass index (BMI), kg/m^2	21.9 (3.6)	21.2 (3.5)	21.1 (3.2)	20.7 (3.1)	20.8 (2.8)	21.7 (5.7)	< 0.001
BMI gain, kg/m^2							
Median	0.10	0.08	0.08	0.12	0.16	0.08	
Interquartile Range							
1st Quartile	−2.51, −0.33	−4.99, -0.34	−5.47, −0.31	−5.15, −0.32	−3.19, −0.28	−0.67, −0.53	
2nd Quartile	−0.32, 0.10	−0.33, 0.08	−0.31, 0.08	−0.32, 0.12	−0.28, 0.14	−0.49, −0.14	
3rd Quartile	0.10, 0.53	0.08, 0.47	0.08, 0.47	0.12, 0.53	0.16, 0.53	0.08, 0.63	
4th Quartile	0.54, 2.98	0.47, 3.79	0.47, 4.04	0.53, 4.62	0.55, 3.35	1.02, 2.43	
Obesity, n (%)	27 (20.6)	105 (13.8)	221 (12.5)	88 (10.6)	13 (9)	3 (20)	0.01
Smokers, n (%)	9 (6.9)	50 (6.6)	127 (7.2)	73 (8.8)	10 (6.9)	1 (6.7)	0.65
Alcohol consumption, ethanol g/week	41.6 (89.4)	32.4 (68.1)	29.8 (61.3)	28.9 (59)	27.3 (53.7)	84.7 (170.9)	< 0.01
Walking hours a week, hours/week	12.8 (12.5)	13.6 (13.2)	12 (11.3)	11.4 (11.6)	11.7 (13.8)	7.9 (5.9)	< 0.01
Shift workers, n (%)	5 (3.8)	37 (4.9)	73 (4.1)	30 (3.6)	7 (4.8)	1 (6.7)	0.85
Depressive symptoms, n (%)	10 (7.6)	48 (6.3)	62 (3.5)	38 (4.6)	1 (0.7)	1 (6.7)	< 0.01

*Data are expressed as the mean (standard deviation) or as the number (percentages).
†BMI gain: difference from BMI followed for a year to BMI at baseline.
‡"Obesity" defined as a body mass index ≥ 25 kg/m^2.

sociation between duration and BMI gain compared with 7-8 h was seen for any sleep duration.

A total of 11,640 participants (11,183 for men, 457 for women) meeting the criteria for obesity at baseline were excluded, leaving 23,212 participants (20,023 for men, 3,189 for women) for multivariable logistic regression analyses. As summarized in Table 3, of 20,023 non-obese men at baseline, 1,171 (5.8%) new cases of obesity were identified. Results of the multivariable logistic regression analysis showed that the risk of developing obesity was increased in those sleeping less than 5 h and 5-6 h compared with those sleeping 7-8 h after adjustment, with odds ratios of 1.91 (95%CI 1.36, 2.67) and 1.50 (95%CI 1.24, 1.80), respectively. Participants who slept ≥ 9 h had an increased OR (1.42), but this was not statistically significant. Of 3,189 non-obese women at baseline, 75 (2.4%) new cases of obesity were identified, but results showed no association between sleep duration and the development of obesity compared with 7-8 h for any duration.

We repeated analyses of the data stratified by age (above/below the median), but the results were found to be essentially unchanged (See Supplementary Tables—available online only at www.journalsleep.org).

Table 2—Prospective association of sleep duration with BMI gain* between 2006 and 2007 in men (n = 31,206) and women (n = 3,646)

	Model 1‡			Model 2§			Model 3¶		
	β coefficient	95%CI	P value	β coefficient	95%CI	P value	β coefficient	95%CI	P value
Sleep Duration (Hours)									
Men									
< 5	0.018	0.035, 0.157	< 0.01	0.017	0.031, 0.153	< 0.01	0.016	0.024, 0.146	< 0.01
5 to < 6	0.014	0.003, 0.063	0.03	0.014	0.003, 0.063	0.03	0.013	0.001, 0.061	0.04
6 to < 7	0.006	−0.012, 0.033	0.35	0.006	−0.012, 0.033	0.34	0.006	−0.011, 0.033	0.33
7 to < 8	0.000			0.000			0.000		
8 to < 9	−0.007	−0.073, 0.018	0.24	−0.007	−0.074, 0.017	0.22	−0.008	−0.075, 0.016	0.20
≥ 9	0.018	0.087, 0.349	< 0.01	0.018	0.083, 0.345	< 0.01	0.018	0.079, 0.340	< 0.01
Women									
< 5	0.009	−0.112, 0.190	0.61	0.010	−0.109, 0.193	0.58	0.010	−0.108, 0.194	0.57
5 to < 6	0.000	−0.080, 0.082	0.98	0.003	−0.075, 0.087	0.89	0.003	−0.075, 0.087	0.88
6 to < 7	0.012	−0.048, 0.087	0.57	0.014	−0.045, 0.090	0.52	0.013	−0.046, 0.089	0.53
7 to < 8	0.000			0.000					
8 to < 9	0.013	−0.088, 0.199	0.45	0.014	−0.086, 0.201	0.43	0.013	−0.087, 0.200	0.44
≥ 9	0.021	−0.153, 0.679	0.22	0.020	−0.164, 0.668	0.23	0.020	−0.163, 0.669	0.23

Abbreviations: 95%CI, 95% confidence interval.
*BMI gain: difference from BMI followed for a year to BMI at baseline.
†Analysis of BMI gain include all participants.
‡Model 1: adjusted for age and BMI at baseline.
§Model 2: adjusted for the variables in model 1 plus shift-worker, smoking, alcohol consumption, and physical activity.
¶Model 3: adjusted for the variables in model 2 plus depressive symptoms.

DISCUSSION

The cross-sectional results at baseline in the present study showed a significant association between short sleep duration of less than 7 h and BMI compared with a sleeping time of 7-8 h among both men and women. Our results were compatible with the previous reports.[10,11] The prospective results of the present study demonstrated that sleep duration < 6 h was significantly associated with weight gain and obesity compared with that of 7-8 h among men. The present study also showed that sleep duration of over 9 h was significantly associated with weight gain. These associations persisted even after adjustment for factors considered a priori to be potential confounders, in our sample of working men. By contrast, the similar prospective associations were not found for women.

Recent longitudinal studies on the relationship between short sleep duration and obesity are inconsistent. Three studies reported an association between short sleep duration and obesity or weight gain.[12,15,17] Hasler et al. reported a strong association between short sleep duration (< 6 h) and obesity, with an odds ratio for sleep duration predicting obesity of 0.50 over a 13-year period.[12] The findings reported may require particular care, since the data were derived from nearly 500 individuals, with the majority having greater psychological symptoms. The large-scale Nurses' Health Study, which followed middle-aged women for 16 years, also reported an association between short sleep duration and increased weight, with relative risks of a 15-kg weight gain for those sleeping 5-6 and 6-7 h of 1.32 (95% CI: 1.19, 1.47) and 1.12 (95% CI: 1.06, 1.19), respectively, and relative risks for incident obesity of 1.15 (95% CI: 1.06, 1.26) and 1.06 (95% CI: 1.01, 1.11).[15] Although these associations were small, they remained significant after adjustment for important covariates. However, these analyses were based on self-reported measures of body weight, and the sample was limited to middle-aged women nurses. The Quebec Family Study showed an elevated risk of weight gain and the development of obesity in short (5-6 h) compared with average duration sleepers (7-8 h).[17] The generalizability of this study, however, may be limited by its small sample size (n = 276) and use of self-reported sleep duration; in particular, the sample may have been predisposed to obesity and weight gain, given that this study was originally conducted to explore the role of genetics in the etiology of obesity. We believe that the present study has provided the strong prospective evidence on obesity associated with shorter sleep duration, at least for men, through improving the limitations mentioned above.

In contrast to these positive findings, three other studies have reported no association between short sleep duration and obesity or weight gain.[13,14,16] Gangwisch et al. reported a negative (β = −0.053) but nonsignificant association between sleep duration and an increase in BMI over 10 years in a sample of approximately 2,500 men and 1,100 women.[13] Bjorkelund et al. showed that for 1,462 women, weight gain was not significantly associated with sleeping < 6 h/24 h on 32 years' follow-up.[14] In the Whitehall II Study with 5 years' follow-up, short sleep duration (≤ 5 h) was not significantly associated with either significant changes in BMI (β = −0.06, 95%CI −0.26, 0.14) or the incidence of obesity (OR = 1.05, 95%CI 0.60, 1.82).[16] These analyses were based on an occupational cohort of approximately 4,000 men and women. No definite explanations can be found at this stage for the discrepancy between the previous

Table 3—Odds ratios of incident obesity* at 1-year follow-up as a function of sleep duration at baseline (2006) in men (n = 20,023) and women (n =3,189)

			Model 1‡			Model 2§			Model 3¶		
	Number (%)	P value	OR	95%CI	P value	OR	95%CI	P value	OR	95%CI	P value
Sleep Duration (Hours)											
Men											
< 5	43 (9.4)	< 0.001	1.96	1.40, 2.74	< 0.001	1.97	1.41, 2.75	< 0.001	1.91	1.36, 2.67	< 0.001
5 to < 6	209 (7.4)	< 0.001	1.50	1.24, 1.80	< 0.001	1.51	1.26, 1.82	< 0.001	1.50	1.25, 1.80	< 0.001
6 to < 7	554 (5.7)	0.13	1.13	0.98, 1.30	0.10	1.14	0.98, 1.32	0.08	1.14	0.98, 1.32	0.08
7 to < 8	294 (5.1)	1.00	1.00			1.00			1.00		
8 to < 9	63 (6.0)	0.20	1.19	0.90, 1.58	0.22	1.17	0.89, 1.56	0.26	1.17	0.88, 1.55	0.27
≥ 9	8 (7.3)	0.29	1.48	0.71, 3.06	0.30	1.46	0.70, 3.03	0.31	1.42	0.69, 2.96	0.34
total	1171 (5.8)										
Women											
< 5	1 (1.0)	0.50	0.36	0.05, 2.73	0.32	0.36	0.05, 2.75	0.33	0.35	0.05, 2.69	0.31
5 to < 6	14 (2.1)	0.86	0.83	0.40, 1.69	0.60	0.83	0.41, 1.70	0.62	0.82	0.40, 1.68	0.59
6 to < 7	40 (2.6)	0.89	1.03	0.59, 1.82	0.91	1.04	0.59, 1.83	0.90	1.04	0.59, 1.84	0.88
7 to < 8	18 (2.4)	1.00	1.00			1.00			1.00		
8 to < 9	2 (1.5)	0.75	0.62	0.14, 2.70	0.52	0.62	0.14, 2.72	0.53	0.64	0.15, 2.79	0.55
≥ 9	0 (0.0)	0.59	0.00		1.00	0.00		1.00	0.00		1.00
total	75 (2.4)										

Abbreviations: OR, odds ratio; 95%CI, 95% confidence interval.
*"Obesity" defined as a body mass index ≥ 25 kg/m^2.
†Analysis of obesity was conducted among participants who were not obese at baseline.
‡Model 1: adjusted for age.
§Model 2: adjusted for the variables in model 1 plus shift-worker, smoking, alcohol consumption, and physical activity.
¶Model 3: adjusted for the variables in model 2 plus depressive symptoms.

data and the present results. Yet it appears that lengthening the follow-up period, e.g., over 5 years, may not necessarily yield the significant association, as seen above. The present findings suggest that future obesity among short sleepers could be observed even at the 1-year follow-up. This hypothesis should be examined in subsequent studies.

The present study found no relation between short sleep duration and weight gain and obesity on 1-year follow-up in women, although the cross-sectional analyses did show a significant association. The lack of prospective association may be related to the low prevalence of obesity and newly developed obesity in these women, at 12.5% and 2.4%, respectively, or to the lower prevalence of obesity in these participants than in the general population. A previous study reported that 19.4% of 26,456 working Japanese women were obese,[22] while the National Government's 2006 National Health and Nutrition Survey reported a prevalence of 21.4% in women aged over 20 years.[3] However, given our cross-sectional findings of a significant association between short sleep duration and obesity in women, further follow-up of the present study would be useful.

Generally, weight gain results from an increase in caloric intake or a decrease in energy expenditure, acting either alone or in combination. Appetite is regulated by the interaction of metabolic and hormonal signals and neural mechanisms. Leptin, an appetite-inhibiting hormone derived from adipocytes, signals satiety to the hypothalamus while ghrelin, an appetite stimulating hormone, is released primarily from the stomach and increases appetite and food intake.[23] Circulating levels of leptin act to counter regulate those of ghrelin, and appear to be markedly increased during sleep.[24,25] In a laboratory study in healthy young men subjected to 6 nights of 4 h in bed followed by 6 nights of 12 h, mean leptin levels were 19% lower during sleep restriction, the nocturnal acrophase occurred 2 h earlier and was 26% lower, and the amplitude of diurnal variation was 20% lower.[26] In a second laboratory study involving 2 days of 4-h bedtimes and 2 days of 10 h, the change in ghrelin-leptin ratio between the two conditions was strongly correlated to the change in hunger ratings.[27] The Wisconsin Sleep Cohort Study, a population-based study involving more than 1000 adults, found that a usual sleep time of 5 h as compared with 8 h was associated with leptin levels 18% lower and ghrelin levels 15% higher after controlling for BMI, suggesting that sleep restriction may increase appetite.[28] Another study involving 740 men and women aged 21 to 64 years found that those who slept 7-8 h had lower indices of adiposity and a lower prevalence of overweight/obesity than those sleeping for 5-6 h, and that leptin levels in those sleeping 5-6 h were significantly lower than that predicted for their total fat mass.[29] This hormonal pattern would be expected to increase appetite, providing a possible explanation for the increased prevalence of overweight associated with short sleep duration.

Orexin (hypocretin) is produced by neurons in the hypothalamus which regulate feeding and wakefulness.[30,31] Orexinergic activity is in turn influenced by both central and peripheral signals, with leptin exerting inhibitory effects and ghrelin promoting further activation.[30,31] This suggests the possibility that the decrease in leptin and increase in ghrelin with short sleep duration might act to increase orexin activity, leading to an increase food intake.

Further, several studies have suggest that short sleep duration might increase caloric intake and decrease energy expenditure via a direct effect on lifestyle activity.[32,33] Ohida et al. reported that sleep loss was associated with a lack of exercise and irregular eating habits,[32] while Stamatakis et al. reported that short sleep duration was associated with obesity-related behaviors such as lower physical activity and lower fruit and vegetable consumption.[33] However, this study showed no significant relationships between sleep duration and exercise in men.

The present study has demonstrated that sleep time of over 9 h has significantly associated with weight gain even after controlling for potential confounders. One plausible explanation for the association between long sleep duration and weight gain is that reduced energy expenditure due to increased time in bed among long sleepers may affect weight gain. The present study however did not show such an effect. Another possibility is that an unrecognized confounder could lead to weight gain and increased need for sleep. An example of such a confounder might be obstructive sleep apnea. Although The Wisconsin Sleep Cohort Study showed longer sleep cause an increase in serum leptin level, sleep apnea may be a cause of leptin resistance and thus a propensity for further weight gain.[28,34] Sleep apnea patients are also reported to be associated with increased proinflammatory cytokines, which contribute to sleepiness.[35] A further possibility is that long sleep could directly lead to an increased risk of weight gain. However, we know of no plausible physiologic explanation for such a cause-effect relationship. Although the mechanisms underlying the association between long sleep duration and obesity are not readily explainable, the present study is consistent with a prior prospective study which showed that long-duration sleepers exhibited a significantly higher increase in body weight than 7-8 h sleepers over 6 years.[17] The generalizability of this study, however, may be limited by its small sample size of our current study.

The present findings showed no significant association between long sleep duration and development of obesity. However, there seemed a reverse J-shaped relationship between sleep duration and the risk of obesity in men (Table 3), with a nonsignificant increase in OR among participants slept 9 h or longer. The similar nonsignificant association has been presented in the previous prospective studies: Hazard ratio of obesity (BMI > 30) 1.03 in women nurses sleeping 9 h or more and OR of being overweight plus obesity (BMI $\geq$ 25) 1.29 in white-collar workers with over 9 h of sleep.[15,16] The unsolved question about the obesity risk of long sleepers requires further follow-up research.

The strength of the present study is its large sample size of approximately 35,000 men and conduct over the relatively short period of 1 year. This short follow-up may have minimized the effects of environmental changes, and thereby improved the reliability of the sleep-obesity association.

Several limitations of the study warrant mention. First, usual sleep duration was self-reported. This should be a continued limitation relevant to sleep epidemiology, but the Nurses' Health Study showed good validity for usual sleep time obtained from a similar question against 1-week sleep diaries ($r = 0.79$).[15] Second, participation was limited to workers in a single company, albeit that the study population was large and the participants were distributed throughout Japan. Third, the number of work hours was not measured. Although the relationships between work hours and weight gain or obesity have reported, it does not seem to be clear the relative contribution of work hours and sleep duration.[36-38] Fourth, physical activity was measured by a self-report of walking time of apparently moderate-intensity activity level. Naturally, this measure cannot cover other kinds of moderate-intensity such as cycling or hard-intensity activities such as exercise and sports. The measurement of physical activity in the present study, however, included daily walking time such as commuting to work, during working on weekdays (workdays) and walking time as regular exercise or recreational exercise and leisure-time of walking time on weekdays (workdays) and weekend (non-workdays); Westerterp and colleagues showed that moderate-intensity activities, not high-intensity activities, influence the total energy expenditure.[39,40] Also previous studies showed the relation between walking time and walking step counts and the relation between walking step counts and net energy cost, which suggested that walking time has a link to net energy cost.[41,42] Therefore, although limited, the measurement of physical activity in the present study could be used to control for its confounding effects. Fifth, calorie intake was not measured. Finally, no adjustment was made for sleep disorders, such as obstructive sleep apnea syndrome. Sleep apnea is presumed play a key role in both sleep disruption and obesity.[43]

In conclusion, we found that short sleep duration was associated with weight gain and the development of obesity over 1 year in men, but not in women.

DISCLOSURE STATEMENT

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